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DISEASES OF THE NERVOUS SYSTEM

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CONTENTS:

BRAIN CONCUSSION: EVALUATION OF THE AFTER EFFECTS BY SPECIAL TESTS (Page 325)
Kurt Goldstein, M.D., Boston, Massachusetts

THE ELECTROCARDIOGRAM OF CURARIZED HUMAN PATIENTS (Page 335)
Arthur Ruskin, M.D.; Jack Ewalt, M.D., *and* George Decherd, M.D., Galveston, Texas

PSYCHOSOMATIC MEDICINE (Page 342)
Solomon Katzenelbogen, M.D., Baltimore, Maryland

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE OF THE CINCINNATI GENERAL HOSPITAL (Page 345)

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Brain Concussion: Evaluation of the After Effects by Special Tests

KURT GOLDSTEIN, M.D.*
Boston, Massachusetts

Brain concussion, without fracture of the skull and without localized cerebral symptoms pointing to gross damage of brain matter, is of frequent occurrence particularly among casualties of war. The concussion may take a varied course. One man may feel recovered quickly, another after some weeks, and neither may show symptoms or deviations discoverable in the usual neurologic or psychiatric examination. Experience has taught, however, that although the patient may intentionally overlook the disturbance and the physician may be deceived as to its severity, these after-effects present, in the case of a member of the armed forces, an important and difficult problem. The physician in charge must determine when to return the soldier to service. This is a decision of utmost responsibility, both to the individual and the service. An individual, who may recover if he is permitted to rest, may be permanently injured if exposed too soon to the physical and mental strains which the service demands but which his organism may not yet be able to endure. A sudden let-down in a situation demanding the man's complete capacities may not only be dangerous to him but may have catastrophic consequences for the military activity in which he is participating.

Experience in the last war has taught that it is impossible to obtain sufficient and correct knowledge of the individual's conditions from the usual neuropsychiatric examination. The problem is enhanced in this war where the demands upon mental and physical capacities are still greater. I tried to apply methods developed during the last war to better judge the after-effects of gunshot brain wounds[4] to this situation. In general these have proved useful. However, the methods must be altered in accordance with the damage. We have to deal with impairment of entire physical and mental capacity, not merely with circumscribed defects as in wounds. Moreover there is no time for a prolonged examination. And lastly it must be kept in mind that the man is useful only if he is able to stand the rigors of service.

The results of tests in the last war were satisfactory. However, no statistical data can be given as we did not start to record the results of the examination in a systematic way long enough before the end of the war.

Attempts to confirm or to correct these results through observation of cases with concussion caused by peacetime accidents, were not fruitful. Concussion from war causes differs in essential points from injuries of peacetime. The physical and mental condition of the individual when the concussion occurs is different. At the front the soldier usually is in a state of abnormal fatigue and strain not encountered among civilians. Even more important are the surrounding conditions which vary to such an extent that the very factors to be considered are different. The civilian will usually be able to avoid efforts he cannot stand, but the soldier will often assay tasks beyond his endurance and capacity. Under these circumstances a careful, detailed

*Clinical Professor of Neurology, Tufts Medical School, Boston, Mass.

examination after brain concussion is indicated. I will outline the procedure I consider useful in these cases.

Acute State of Brain Concussion

Acute states of brain concussion show great differences in severity. A symptom in all, however, is change in consciousness. Some kind of disturbance of consciousness almost always accompanies a real concussion of the brain. The disturbance may vary greatly in degree and duration. Sometimes loss of consciousness is brief or there was no opportunity of observing, and the man himself has amnesia for it. We can conclude that there was disturbance of consciousness from the amnesia the individual shows for the accident and the time shortly before or afterwards. A man who shows real amnesia (See seq, page 3 s q) was without doubt, unconscious. Consciousness is not a clear concept. There has been a disturbance of consciousness when the normal reactions of the individual to events in his environment are diminished or when there is inability to remember events during that period. We determine the degree of disturbance of consciousness from the variety of the reactions or from the memory defects.

The nomenclature by which various degrees of consciousness is described is confused and confusing. Often the same term will be used by one physician to indicate a certain condition and by another to describe something different. It is frequently difficult to get a definite idea about the earlier condition from his report. It is to be welcomed therefore that a committee of the Medical Research Council in England has set forth definitions of various states of consciousness which in my opinion bring out clearly characteristic differences and conduce to better understanding by the physician and better description in his report. *Coma* is defined as a condition of absolute unconsciousness, absence of any psychologically understandable responses to external stimuli or inner needs. Some physiological reactions such as reflexes, etc., may or may not be present; their lack is not a criterion for the assumption of coma. *Semicoma* is a condition where psychologically understandable responses are elicitable but only by painful or other disagreeable stimuli. *Clouded consciousness or confusion,* is characterized by impaired capacity to think clearly and with customary rapidity, to perceive, respond to, and remember current stimuli. Here the patient is reacting correctly to many stimuli, but his reactions may change. At one time he may react promptly and correctly, then not at all or in a wrong way. This change of condition may concern all performance fields. He is most frequently disoriented as to time, space or person. Confusion is often accompanied by delirium and motor restlessness. *Stupor* is lack of activity (he does not speak, he does not move). Various degrees of confusion can be distinguished by noting the extent to which the man reacts to demands.

The degree of intensity of disturbance of consciousness can in general be considered an indication of the severity of the brain damage and is therefore important to formulate an opinion of the patient's condition and prognosis. However, there is no definite parallelism. Of even greater importance, is the *duration* of the change in consciousness. Indeed, return to consciousness must not be considered evidence of recovery of the brain damage. We must try to find objective criteria for the determination of the degree of damage to the brain. The neurological and other clinical symptoms give some hint. During the condition of consciousness there may be changes in reaction to light or dilated pupils; the patellar reflexes may be absent and a flaccid paresis is present. These and other neurological symptoms may disappear early. Unfortunately, we have few useful neurological symptoms after consciousness has returned. Deviations in temperature and pulse are important. Persistent subnormal temperature with rising pulse indicates a bad prognosis. If the temperature rises with the pulse, the prognosis is more favorable, particularly if the patient shows at the same time, signs of weakening from unconsciousness. Increased temperature may indicate some localized or infectious process, i.e., subdural hematoma, meningitis, etc., but it is not a sign of shock. On the contrary it is a sign that the general shock has probably ceased. This is a condition where surgical intervention may be indicated.

There is another phenomenon in pulse which deserves attention. The difference in the pulse of a normal person in reclining, sitting and standing positions is not great, usually not more than 5 to 10 beats a minute. In

brain concussion one frequently finds a wider margin. However, that also may disappear. If the patient shows a great variation after having presented a small range for some days after the injury, one must be on the lookout. It may be a sign that the total condition is getting worse.

Even when lumbar punctures are made the findings are not uniform. In some cases we may find increase of intracranial pressure, in others none at all. As Denny Brown has pointed out: There is no direct relation between the increase of pressure and the degree of unconsciousness. Therefore, we should not expect a lumbar puncture to cause changes of consciousness to disappear. It is doubtful whether a lumbar puncture should be made in the acute case. I think it should be done only if there are definite symptoms or intracranial pressure.

It might be argued that electroencephalography and pneumoencephalography could give hints as to the degree of damage to the brain. Dennis Williams,[13] who is responsible for careful investigations in the use of the electroencephalogram in head injuries, reported that directly after brain concussion there are slow waves and changes of voltage. Sometimes there is a reduction of voltage; at others, waves of high voltage occur as in epilepsy. I would like to add here that even cases which show the spikes do not necessarily suffer later from epileptic attacks. In the confused state the waves become irregular with some lightening of consciousness. The slow waves are mixed with quicker waves and the latter increase until a normal condition returns. The amount of generalized abnormalities is proportional to the severity of the injury and gradually diminishes with recovery. Cases in which normal records are made from the very beginning or occur earlier are prognostically more benign. However, the abnormalities may disappear and clinical symptoms may persist which we must consider as organic sequelae of concussion. Thus in a stage where the electroencephalogram should be particularly helpful in diagnosis, it is not. Negative or positive findings in the electroencephalogram do not exclude the possibility of organic damage or call for a diagnosis of neuroses or of malingering.

Denny Brown and Russell[3] concluded from experiments with cats that brain concussion is due not to gross damage of the brain but to an immediate paralysis which is a "direct traumatic central inactivity." The characteristic changes of the E. E. G. is due to this central inacivity. It is closer related to the symptoms observable in the acute and subacute states than in the after effects.

Pneumencephalography is no more useful in diagnosing of concussion. There can be positive findings in the encephalogram but the concussion may be slight or absent. With encephalography we recognize certain circumscribed defects but not the concussion. That may happen as Denny-Brown writes, when penetration occurs and the skull may show multiple fragments. On the other hand we may find nothing in the encephalogram where there is no doubt that there is a brain concussion.

Thus, since electroencephalography and encephalography do not assist us in diagnosing the nature of the condition our judgment must be based on the consideration of the entire physical and mental condition. That is true particularly in arriving at a decision whether the man should be returned to service.

If the patient does not recover consciousness within two or three hours, we know we have a severe case on our hands. Immediate treatment is imperative because of the danger to life. All movements must be avoided. Primary attention must be directed to treatment and prevention of surgical shock, which such patients frequently show. If the patient has weathered the initial days, he may recover, even though there were very severe symptoms at the outset. The recovery may go through the same stages characteristic of a condition which from the beginning had been less severe.

In striking contrast to these severe cases are those in which early disturbances are slight. The individual may have thought the accident so trivial that he continued his activities. He may not remember how long he was unconscious, if at all. Directly after the injury flaccid paralysis with loss of reflexes may have occurred concommittently with a brief disturbance of consciousness. It lasted for such a short time that the man did not remember or recognize the disturbance. But he feels dizzy, has headaches, may show vomiting, may be somewhat confused, restless, excited and may have amnesia for the injury. After some

hours or days, he may feel well except that he is weary and has occasional headaches. Even after such slight incidents, symptoms may appear days later similar to those in the more severe cases, and the after effects of some of these slight injuries may be serious without our being able to assign a reason.

Between these two extreme conditions there are a multitude of cases where after a longer or shorter stage of unconsciousness many symptoms are present. These, in addition to the disturbance of consciousness, are the characteristic symptoms of the acute stage of brain concussion.[12] There may be coma, stupor, abnormal excitement and restlessness; the patient may be able to answer questions correctly at one time and later not at all. There is wide fluctuation in mental condition. There may be incontinence, vomiting, delirious episodes. After some hours or days the man becomes clearer, improves in his contact with the outer world, and shows diminishing deviations in his behavior. However, more careful observation frequently reveals definite mental changes—disorientation as to time, space, persons, lack of memory, defects in judgment, confabulation—a picture similar to that in Korsakoff's psychosis. Recovery from this stage may occur in hours or days or sometimes in weeks. It is never certain that the patient will not relapse into a more severe state.

In these various mental conditions there are parallel physical deviations. Gross neurological symptoms do not play a determining role in the picture if there is no complication of a gross circumscribed defect in the brain. During unconsciousness there may be sluggishness or lack of tendon reflexes, a positive Babinski, etc. Similar anomalies of temperature and pulse, mentioned above in connection with severe damage may be observed, occurring for a relatively short time, possibly hours or a few days.

Clinical Symptoms in the Period After the Acute State

After the disappearance of recognizable cerebral symptoms the patients of this group may not have definite complaints at all. They may feel generally recovered but in spite of this they may show amnesia for a long time. This may continue for two months or longer, and many indefinite subjective symptoms as headache, dizziness, and emotional instability may be present. Finally, deviations which can only be ascertained in special examinations come to the fore. For a correct evaluation of the man's capacity for service it is necessary to observe these symptoms very carefully. That holds true also for the men who have shown trivial symptoms from the outset. Headache is very common. It is described by the patient as a throbbing, or hammering or a feeling of pressure, etc. The symptoms may be continuous or attacks may occur at longer or shorter intervals. The patient may be hyper-sensitive to noise, light, effort, and when exposed headache may increase. The headache seems to be particularly severe in cases where there were severe symptoms at the onset. Dizziness occurs spontaneously or upon sudden changes of the position of the body or the head. The patient experiences a sensation either that the world is turning around him or uncertainty as to his own body. Under these conditions changes of the experimental excitability of the labyrinth are not always in evidence. Dizziness is usually accompanied by vasomotor disturbances and nausea, particularly if it occurs during attacks. The emotional instability expresses itself in abnormal irritability, sensitivity upon physical or mental effort, anxiety, depression, etc.

Even if in the usual clinical examination no objective symptoms are found, it is difficult as Doctor Symonds, a captain in the Royal Air Force, observed, "for any one who has studied a large number of these cases to escape the conclusion that the organic factor is present." However, the suspicion of neurotic reaction reasonably arises especially if one sees the patient for the first time some weeks after the concussion. It would be a factor of prime importance if we could find in the symptomatology, definite objective criteria for distinction between organic after effects and neurotic reactions. In severe conditions differentiation is not diffifult. An extreme tubular form of the visual field or certain types of disturbances of sensation or motility make the diagnosis of neurosis easy and certain. A positive Babinski, lacking reflexes points definitely to an organic defect. The onset of amnesia, not directly after the injury, but after an interval or if it can be removed by amytal or other hypnotics indicates a neu-

rotic amnesia. Amnesia concerning the injury and the time directly afterwards (real amnesia) is a symptom leaning toward organic defect.

However, the evaluation of some symptoms as neurotic or organic does not exclude the possibility that the individual is suffering from organic damage in addition to neurotic reactions or vice versa. A decision in these cases is not easy because the symptoms are not exclusive. The same symptoms may be present in organic and neurotic conditions. It is difficult to conclude from general impressions because the soldier usually minimizes the symptoms whereas the civilian in peacetime is likely to exaggerate. The soldier dissimulates; he is anxious to lessen his subjective disturbances; he is eager to return to service. On the other hand correct evaluation is imperative since responsibility for the decision is great, both to the individual and the service.

Significant as differentiation between organic and functional symptoms in postconcussion condition is, of itself it is not decisive of the question concerning the capacities of the man in service. The consideration of symptoms in relation to the demands of the service is of great consequence. So-called "functional" disturbances of equilibrium or vaso-motility, etc., are equally serious handicaps. Of course if we could say with certainty that a symptom is organic we could estimate more accurately its significance in the duties of service. Therefore our efforts to find methods which aid in such a decision must be unrelenting. Of equal importance is the need to eliminate the real malingerer, whose number fortunately is not great.

Examination of Patients in the Postconcussion State by Means of Special Tests

To discover men who are likely to be hurt if they return to service, I have selected a number of tests which are helpful in determining the doctors' recommendations. The tests have been developed from symptoms observed in men with brain concussion and are used in addition to the ordinary clinical methods. The examination differs from the usual clinical examination so that symptoms are more easily and more definitely detected. This is due to a finer differentiation of tasks and a more detailed evaluation of results. I was influenced in my procedure by the knowledge that a slight diffuse damage of the brain cortex may not produce such gross changes that will be revealed by the usual clinical methods. The existing changes may be very slight affecting only finer performances of higher mental capacities and require more delicate methods to uncover them. Damage to these more complicated functions must not be overlooked because incapacitation may be especially detrimental in the activities of the man in service.

Examination of the following performance fields is of particular significance: the vaso-motor system, the visual sphere, the sphere of equilibrium and of the higher mental capacities. I have compiled a series of tests consisting of tasks in these fields. It would excessively enlarge this preliminary note if I were to give a detailed description of all the tests and discuss their results. Furthermore, there are insufficient records at hand to lay down hard and fast rules. It is my purpose here to induce research along these lines. These methods are set forth in detail in my recently published book, "The After Effects of Brain Injuries in War."

Nevertheless, it is necessary to touch upon the general character of these tests. Application is simple, requires little time and can be used in routine examinations. Some have to do with physical phenomena; others with mental. In regard to the latter we do not attempt to gauge capacity in a statistical evaluation, but try to determine: (A) How the man behaves in performances which in themselves are familiar to him and easy to fulfill, but which he has to continue for a longer time or to execute at a faster rate. Continuation of a familiar task for a longer time and at a predetermined speed is characteristic of the tasks set for the man in the service. (B) In a second group of tests the task demands some special mental capacity; to make decisions or to alter them. This capacity is particularly important in military service.

I. Disturbances in the vaso-motor sphere are frequent in the post concussion state. The pulse may at one time be abnormally low; at another abnormally high. Characteristic is the change of pulse rate under the influence of special positions and movements of the body, and of mental excitation. If, for instance, the pulse is noted in reclining, sitting

and standing positions, or after 10 knee bends, etc., identifying changes of the pulse rate may occur. While similar changes may be found in persons not suffering from concussion, neurotics for instance, it is imperative that we discover any neurotic symptom. These tendencies, no matter what their extent or cause, should be revealed if they exist in the soldier. Pulse rate changes are almost always present in the vaso-motor disturbances. In our patients vaso-motor irritability is usually more noticeable. If this symptom disappears after a few weeks, it is a good sign. On the other hand a recurrence after an interval should be investigated fully. A pulse difference in reclining and sitting positions greater than 20 beats indicates abnormal irritability. The same is true if the increase of the pulse after 10 knee bends of about 20-30 beats does not return to normal in approximately one-half minute. Outstanding vaso-motor irritability should be given careful consideration before sending the man back to duty. This disturbance may cause physical or emotional failure.

II. Disturbances of equilibrium are frequent after concussion. They are produced by lesions of the vestibulum or the cerebellum both of which are often affected by brain concussion. Subjectively the man complains of headache, dizziness, uncertainty in performances which demand sense of balance. The excitability of the labryinths may not be changed, but sometimes abnormal sensitivity to caloric stimulation is found. Existing disturbances may easily be overlooked, because they are not observed while the patient keeps his eyes open.

Simple tests such as the following should be included in each routine examination: Standing with open and then with closed eyes; arms stretched forward, and sideward; walking forward and backward about 6-10 meters with open and with closed eyes; pointing to the nose with closed eyes; finger-to-finger pointing with closed eyes. These tests while simple to apply, unfortunately are not as easily evaluated. There are many possibilities which may effect the results, such as unintentional interferences by the subject, without the examiner realizing it. Only intimate acquaintance with these tests will prevent deception and render them useful.[5,6] I mention a few factors which must be looked for: In standing or sitting, the legs of the subject should remain closely parallel, the head straight and the eyes always directed straight ahead. The man's attention should not be distracted. In the finger-to-finger pointing test the stimulus should be presented at such a place that the man has to move his arm in a lateral plane. If attention is paid to these and other significant factors, one can detect uncertainty of movements, deviation, past-pointing, falling, etc. As long as such deviations from the norm are present the man should not be returned to service.

III. The usual eye examination may show no pathology as there may be no gross defects in the visual field. Concentric constriction has often been considered a neurotic sign, but experience with patients suffering from gross brain injuries demonstrates that this condition is not a dependable criterion. The constriction may occur after brain injury or concussion particularly if there is diffuse damage of function in the brain matter. It is important practically that the constriction shows definite differences from that in neurotics.[7] It especially affects the temporal parts of the field and it may occur in the form of a bitemporal constriction. It never reaches the high degree found in neurotics. The border line of the field runs about parallel to the normal border, while in neurotics the line is circular about the center. If an area of tubular field is evident it may be a sign of organic damage; in testing the field at great distances it is not as enlarged as we would expect judging by the physical condition. Indeed, extended tubular field is characteristic of neurosis, but we should not consider every kind of abnormal constriction at greater distances as neurotic. Taking into consideration the fine differences between field defects in neurosis and in organic condition, the constriction will be a significant symptom. It is an expression of abnormal fatigability and should be taken seriously into consideration.

Another sign of abnormal fatigability to be sought in the visual sphere is the annular or ringscotomata.[8] With the usual perimetric procedure, the outer border of the field is determined either by proceeding from the center outward, or from the periphery inward. The object is usually moved only to the point where it disappears from or just comes into the field of vision. By this method a shrinkage of the field may be detected. If, however, after fixing the outer border, the test object is moved

toward the center along the same meridian, it may be observed that the subject now does not see the object at the place he located it before. On continuing the procedure, once he may see the object then he does not see it and so on. Usually there is no defect at the center. Proceeding in this manner along all meridians, similarly located zones of perceptual unresponsiveness will be found. If then the zones of better vision and of poorer vision are connected in the usual mapping-out method, concentric rings of better and poorer perception (the so-called Ringscotoma) will be recorded.

These annular scotomata are not the sequelae to damage of the retina or the calcarina in annular form, but to a defect in the functional excitability caused by abnormal fatigability in the visual sphere that is organic in etiology. The scotomata are the result of two factors: First, the normal difference in excitability of the central and peripheral parts of the retina, i.e., decrease in excitability from the center to the periphery. Second, abnormal fatigability of the entire visual sphere. The latter gives expression in the following phenomena: If we start at a peripheral point with a stimulus strong enough to excite at the onset the stimulation gradually fails owing to fatigue. Because this fatigability also concerns the other retina the same stimuli may be inadequate to stimulate at a point closer to the center, so that we find a blind spot there. Still nearer to the center, the normal increase of excitability may be greater than the decrease by fatigue, therefore the stimulus is perceived. If we proceed centripetally without interruption, we may reach a point where increasing fatigue again overcomes the improvement due to the greater sensitivity, and again we have a scotoma. Since in the center, normal excitability is usually strong enough to respond to a stimulus in spite of fatigue, perception is not disturbed. In moving the stimulus from the center to the periphery, we soon reach a part of the retina where the visual system no longer responds to stimulation at all, in this procedure both factors—fatigue and normal decrease in excitability—become effective. Thus we have a more constricted field if we carry the test from the perisphere to the center. Differences in visual fields in centripetal and centrifugal perimetry also occur when we lengthen or shorten the time intervals between stimulations, producing the annual scotomata. This is a marked example of the great lability of the threshold characteristic of all disfunction of the cortex, and also where there has been a concussion. Because this abnormal lability of the threshold handicaps the patient, the demonstration of annular scotomata is important in our judgment of the man's capacity. As long as the man reacts in this manner he needs further rest and should not be sent to service.

IV. *Testing of disturbances in the mental sphere.* The changes here which are particularly significant determining the usefulness of the man in military service are: (A) *Diminution of the physical and mental performance capacity in general,* measured by the occurrence of fatigue on continuous work; and (B) *Diminution of higher mental processes.* In both respects the use of special tests will reveal facts on which an opinion may be based with a degree of correctness. From a number of tests applicable for this purpose the following are recommended because of their simplicity and relative reliability.

1) *Continuous addition test.*[4] A sheet of paper is given to the subject with digits arranged in vertical rows. The subject is asked to write the sum of two succeeding numbers alongside these numbers. The individual additions are so simple that they can be accomplished without difficulty. The individual continues this addition for one-half to one hour. If he cannot continue, he is permitted to stop sooner. At minute intervals the examiner checks the sums completed by the patient, being careful not to disturb him. The results are recorded as to number of correct and incorrect answers per minute, and the total number of additions is plotted against the number of minutes. On the basis of this graph we can form a conclusion as to the individual's general performance capacity. Output is indicated in the number of problems completed per minute by height of curve; capacity for adjustment in the first part of the curve; capacity to keep up continuous effort in the length of the curve, accuracy and constancy during continued work, in the form of the curve. Deviations—lengthening of the period of adjustment, fluctuations during the period of plateau, precipitous lowering and early end

of the curve—may be considered signs that the brain condition is not normal.

Errors in these simple sums should not occur with people of average intelligence and therefore, if there are mistakes, they have significance which should be analyzed. The test should be repeated every week or two as long as a curve shows any identifying deviation. Improvement is partially due to exercise, which will not, however, mask real defects.

The ergographic test checks continuous performance of a simple physical task. It reveals the same deviation of performance as the addition test.[4]

2) *Reaction test:*[4] This gives insight into the fatigability of the subject as well as the deficiency of higher mental processes. In this test the subject presses a key on the sudden appearance of a noise or light stimulus. The normal reaction time to a sound stimulus is 120-130 sigmas; to light, 170-190 sigmas. About thirty of such reactions are recorded in one series. A normal individual shows an almost horizontal curve in this test during the period of about thirty trials. Abnormalities present themselves in unusually long reaction time, in fluctuations of reaction time and in an early increase in required time. These are portents that the man's capacity may break down under stress. Deviations from normal are still more apparent if the subject has to make a choice in addition to performing a simple operation. Two different stimuli are presented; e.g., a large and a small light, or two differently colored lights. The subject is instructed to press a key on the appearance of the large light, and another key on the appearance of the small one. The normal individual after a short period of adjustment has no difficulty in making the correct selections in thirty trials. The total reaction time is compared to that in the single stimulus plane, 350-450 sigmata. Deviations from the normal curve in the single reaction test indicate abnormal fatigability. If the subject shows normal behavior in the single reaction test but definite deviation in the choice reaction test, both as to protracted reaction time and fluctuation with more or less errors, it is an indication of impairment of the higher mental capacities which are called upon in performing the complicated duties of the service.

3) *Abstraction test.*[9] The usual intelligence test is not adequate to gauge the soldier's capacity. For this purpose special tests have been arranged. A normal person exhibits two attitudes toward the world: The concrete and the abstract. In the concrete attitude we experience and recognize a given thing or situation immediately. Our thinking and acting are directly determined by the present claims upon us. In the abstract attitude we go beyond the current claims of objects or of sense impressions. Specific properties or situations are overlooked. We are oriented in our actions by a conceptual point of view which takes into consideration the demands of the entire situation. The abstract attitude influences such abilities as: (1) Acting voluntarily; (2) shifting knowingly from one aspect of a situation to another, i.e., making a choice; (3) keeping in mind simultaneously various aspects of a situation; (4) grasping the essential parts of a given whole, breaking up a given whole into component parts and reacting to them voluntarily. The normal individual combines both attitudes and is able to change readily from one to the other, according to the demands of the situation.

It is scarcely necessary to emphasize the significance that the loss of the abstract capacity would have on military activities where rapid and purposeful shifting from one aspect of a situation to another is vitally important. Since the presence or reduction in abstract attitude may be determined by tests, we are able to conclude from the findings whether the post-concussion patient is potentially capable of adjusting himself to the requirements of service.

Several tests have been worked out for this purpose.[9] One in particular that is recommended is the Cube Test. The subject is requested to duplicate simple color designs with blocks. Each block is of the same size as the design and is colored on all six sides, corresponding to the colors used in the designs—blue, red, yellow, or white (one color sides) on four sides; the two remaining sides bear two colors separated by a diagonal line, one side being blue-yellow, and the other white-red (two-color sides). In copying the printed design, the patient uses four blocks. The reproduction will be four times as large as the model.

Analysis has revealed that difficulties in

performance arise mainly because of the following factors:

a) Difference in size between the whole design and the four blocks. The designs are one fourth as large as the subject's reproductions. That this is the reason for the patient's failure is demonstrated when he proceeds correctly with a design, corresponding to the area of all four blocks.

b) Inability to make the divisions which are necessary for duplicating the design with four blocks. The subject has to disregard the figure and dismember the design in new mosaic elements. This division needs "abstraction."[9] That the incapacity to make this division is a cause of failure is shown by the fact that if the design is presented with lines drawn dividing it into four squares, the patient may succeed.

c) If the impairment is very great, then even this division by lines may not be sufficient to help. In such cases, the patient may only be able to succeed if he is presented with an actual model made up of four exact duplicates of the blocks used in solving the problem, and then asked to copy this model. Observation on organic patients showed that these different presentations of the design, and finally, the model itself, present a graded scale of difficulty. This gradation in difficulty is useful for testing the degree of impairment of the abstract capacity. Therefore, we present the test in the following steps of decreasing difficulty:

1) The standard card design, that of the small size.
2) The enlarged design without lines.
3) The lined small design.
4) The lined enlarged design.
5) The blocks.

Whenever the subject fails in one of these steps he is presented with the next easier step. Whenever he succeeds in one he is given the standard design again, to determine whether he has learned by this help.

Deviations from normal are manifested by:

A) Exceptionally prolonged solution time, and increase in number of trials before success on each design. However, if the patient is finally successful without assistance, length of solution time or increase in number of trials does not necessarily indicate pathological change, but the latter should be suspected. If the patient is better able to reproduce another design, the protracted solution time on the first test may mean little. Normal persons may start to duplicate total figures or the upper or lower parts of the design, or they may begin by building two figure parts of the corners, thereby losing time. Normal individuals soon see that they cannot reach a correct solution in this way; or if they do arrive accidentally at a correct solution, they will recognize that it is more expedient to proceed by the simpler division method. If the subject persists in proceeding in this total or subwhole manner, even after the other procedure has been demonstrated to him, impairment of abstraction is indicated, even if he should eventually succeed.

B) If the subject was successful in constructing a larger design or one with lines, and thereafter is unable to do the small unlined standard design, impairment of abstraction is definitely indicated. The same is the case if he is unable to duplicate the next standard design after he had been previously successful on a large design or a design with lines. The test may be repeated after certain intervals to bring out changes in behavior indicating progress in capacity.

C) A person with good capacity for visualization may reach a correct solution simply on the basis of imagery, in spite of a persistent defect in abstraction. His handicap will come to the fore if he is confronted with a design he has never seen before. The most important point is whether the subject is able to *learn the piecemeal procedure.* If not it indicates *impairment of abstraction.*

5) For a quick survey of mental capacities, tachistoscopic examination can be of great value.[4] The idea in this examination is to exhibit figures or other objects for a short time (1/5 or 1/10 of a second) and have the subject report what he has seen.

The simplest procedure is as follows: We project the material on an opaque glass screen (about 3 ft. by 3 ft.). In the center of the screen is a barely visible black point. The patient is instructed to locate this point, and later an object will appear for the subject to identify. His attention is aroused by calling "Attention" before the object appears. A simple projection apparatus with a shutter that can be regulated will serve the purpose. After the presentation the patient is required to tell what he has seen or make a drawing of it on

a piece of paper. The object presented, the presentation time, and the verbal reaction of the patient are recorded.

The rationale in using a tachistoscope is that even in normal psychology, perception requires time. In "reduced" time we first perceive the general character of the object presented ("something big, black," etc.). From this general impression we proceed to more and more detailed perception. We know that, for example, familiar objects need 1/10 second of presentation to be recognized, as do simple geometric figures or designs. Experience with patients with brain damage reveals that while they may behave normally under the usual continuous presentation of objects, they show definite defects if presentation time is shortened to a point which would not disturb a normal subject in any way. Thus patients may be unable to read or recognize an object in .2 or .3 of a second, which a normal person would perceive in .1 of a second. By this means disturbances may be disclosed which would escape detection when investigated in the usual manner. If the patient behaved normally at the beginning, but after a short while is not able to proceed or if he shows deviations from normal behavior, excessive fatigability is demonstrated.

Studies made by the Rorschach blots on patients with brain concussion corroborated our results. As characteristic of deviation from the normal, Brussel, Grassi, and Melniker[1] found changes which are similar to those found in our test. The Rorschach test reveals abnormal concreteness and lack of abstraction. "The patient's productions are," the authors conclude, "dictated entirely by the immediate situation so that they are unable to discriminate between the essential and non-essential. Their naming of a blot represents conceptual thinking only superficially, and they are unable to reorganize or evaluate the specific stimulus situation in terms of their concepts."

As specific failures corresponding exactly to those in our tests, I mention the following: Intense efforts toward precise reproduction; automatic color matching; total indiscriminate use of the block to include every detail; attempt to include details irrelevant to the concept, losing relation to the whole, etc. This similarity confirms our finding. The Rorschach may be incorporated in the series of tests. However, the use of the Rorschach test requires considerable experience in administering and in evaluating the responses.

Not all the tests described here for determining the capacity status of a man who suffered from a brain concussion have the same diagnostic value. However, if he fails in several of them he should not be discharged as recovered. Of particular significance are failures in the tests of abstraction and the vasomotor disturbances if they reach a high degree.

I am fully aware that the use of these tests may not be conclusive in the decision to return the individual to service. I consider this battery of tests a framework for orientation, and an outline of the methods of testing to be performed in these cases. As I said before, my experiences were not in sufficient number to permit absolute conclusions. However, even in the form presented this test battery is a comprehensive starting point for further studies.

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The Electrocardiogram of Curarized Human Patients*

ARTHUR RUSKIN, M. D., JACK EWALT, M.D., *and* GEORGE DECHERD, M.D.
Galveston, Texas

A purified, standardized extract of curare** has been used to prevent fractures during the convulsive treatment of certain psychotic states and as a means of temporary relief of spasticity in some dysfunctions of the nervous system. Adequate curare dosage decreases in a very satisfactory manner the severity of the convulsive reaction induced by either metrazol or electricity. Many clinicians have not used this protective agent because they fear the physiologic action of the curare on the respiratory and cardiovascular functions. Physicians who use curare routinely have been uncertain as to the advisability of its use in those patients with cardiac dysfunction, who need treatment so urgently that convulsive therapy seems warranted in spite of the hazards introduced by the circulatory disturbance. Cardiovascular and electrocardiographic studies have been made in several series of patients subjected to shock therapy by insulin, metrazol, and electroshock.[1] At this time it seemed desirable to study the effect of curare on the cardiac mechanism and function, particularly to determine whether or not there were dangers associated with its use in individuals with cardiac disease.

The pharmacology of various forms of curare has been studied for many decades. Curine, an alkaloid obtained from tubocurare (bamboo tubes) and for which an isoquinoline base has been postulated, has been shown to have marked cardiovascular effects. It was studied as early as 1890 by Tillie.[2] A recent study and review by Hauschild[3] showed the production of auricular and ventricular premature beats, and vasoconstriction, in the frog. In the cat and rabbit, however, the blood pressure was apparently unaffected until ventricular standstill led to death; neither acetylcholine nor atropine interfered with these effects.

Most authorities mention few if any cardiovascular effects of the other alkaloids of curare, the most typical of which is curarine obtained from Strychnos toxifera. Goodman and Gilman[4] state that cardiac failure occurs only indirectly from the anoxia or asphyxia of respiratory arrest. Sollmann[5] states that, as compared with the effect on the motor endplate, the heart is not affected except with much larger doses. In other words, the nicotine-like depression of the vagus is masked in the usual dosage, just as is the strychnine-like effect on the spinal cord. Nevertheless, Tillie demonstrated that under artificial respiration a fall in blood pressure occurs in animals. This is presumably due to paralysis of the peripheral sympathetic ganglia with resulting vasodilatation. Dale and Laidlaw in 1911 demonstrated an increase in heart rate due to vagus ganglia depression.[5] The above circulatory effects last only fifteen minutes.

Bennett[6a] found no circulatory effects when curare was used in conjunction with metrazol shock therapy in man. Harvey and Marsland,[7] in a pharmacological study, mention a slight fall in systolic pressure and slight rise in pulse rate, with no change in cardiac output measured ballistocardiographically.

Of the analogues of curare, quinine ethochloride and methochloride, and beta-erythroidine hydrochloride have similar, though weaker, effects on the neuromuscular synapse in man. Quinine methochloride produced the most marked fall in blood pressure and rise in pulse rate, as compared to erythroidine and "unauthenticated curare;" only erythroidine caused a slight fall in cardiac output.[7] Other studies[8] have shown beta-erythroidine to cause marked parasympathetic effects in animals, e.g., cardiac slowing, which could be prevented by atropine or vagotomy. This has even been thought to be a factor in death from supposed heart failure of one human case.[10]

Other analogues of curare are ammonium bases and quaternary ammonium salts, amides, amines, choline, muscarine, methyl strychnine, pyridine, quinoline, piperidine, guanidine, snake venoms, botulinus toxin, the nicotine series, and certain products of muscle metabolism.[6c,7] Some of these have been shown to have cardiovascular and electrocardiographic effects.

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*From the Departments of Medicine and Neuro-Psychiatry, University of Texas School of Medicine.

**Intocostrin, Squibb.

The known actions of "unauthenticated curare" in man can be summarized as follows.[6,7,10,11] The intravenous injection of one to two milligrams per kilo., accomplished within 60 to 90 seconds produces skeletal muscle flaccidity in the following order: ptosis and inability to raise the head, weakness, followed by paralysis of the arms, legs and finally the muscles of the trunk. Diplopia and mask-like facies occur in some cases. These effects begin in approximately 2 minutes and last from fifteen to twenty minutes. Overdosage or rapid injection produces paralysis of the respiratory muscles, including the diaphragm. Before severe respiratory difficulty develops, dysarthria and dysphagia, due to palatal and pharyngeal palsies may appear, and mild inspiratory dyspnea may be observed. Adrenalin has been reported[7] to relieve the dyspnea, but this lacks confirmation.

Prostigmine, potassium and guanidine are known to act as antagonists to curare.[12] In clinical work 0.5 to 1.0 mgm. of prostigmine hydrochloride given intravenously will abolish the curare effect within a few minutes and allow the patient to resume normal respiration. Artificial respiration and oxygen insufflation are of prime importance in the emergency management of respiratory muscle paralysis. Asphyxia is reported to hasten the recovery of the vagus pathways paralyzed by curare in animals, but prostigmine is most effective in restoring vagal function.[13] Eserine also relieves the neuromuscular effect of curare.[14]

The nature of the neuromuscular block produced by curare is not completely understood. Analogies have been drawn with myasthenia gravis, since both conditions are characterized by flaccidity associated with low voltage electromyograms, and both are relieved by the same drugs.[12] Paradoxically, serum cholinesterase is inhibited by curare[15] while the amount of acetylcholine at the myoneural junction is not affected. It is therefore to be inferred that curare acts by raising the threshold of muscle responsiveness to the normal concentration of acetylcholine[12,16] or by lowering the endplate potential.[17]

Theoretically the same action might be anticipated at the vagus nerve endings in the heart. In an animal study,[13] curarization led to no electrocardiographic changes. However, the usual cardiac manifestations of vagal stimulation were prevented because of the resulting vagal block. One report[16] mentions "irreversible" electrocardiographic changes, ascribed chiefly to the apnea which occurred after curare plus electric shock therapy. It is well known, however, that all varieties of shock therapy may themselves result in electrocardiographic alterations, which are usually minor.[1] We have found no publication on the effect of curare on the human electrocardiogram.

Vagus stimulation is known to modify the rate and site of rhythmic stimulus formation in the pacemaker of the heart. Conduction through both the atrium and the atrioventricular junctional tissue is also affected. It may likewise produce changes in the amplitude and direction of the T-waves and in the duration of the Q-T interval. Digitalis is thought to act in part by vagal stimulation. Hence vagal inhibition by curare might conceivably produce electrocardiographic changes, particularly in patients who are digitalized, who have conduction defects or shift in the site of the pacemaker, or who have atrial circus mechanisms in operation. We have studied patients exhibiting all of these disturbances.

Method—We began by taking electrocardiograms in connection with the use of curare for relieving spasticity in some neurological patients. Our first cases (Table I) were cases of familial spastic spinal paralysis and transverse myelitis with little or no heart disease. We then deliberately studied patients with more and more severe grades of cardiac disease including those with electrocardiographic evidence of myocardial damage, congestive and anginal failure, various arrhythmias, and even myocardial infarction.

A preliminary five lead (I, II, III, C $F_{\underline{2}}$, and IV F) electrocardiogram was taken. Then the calculated dose of curare, usually 1 to 1.6 milligrams per kilo., was injected intravenously in a period of approximately sixty seconds. Immediately afterwards, and at frequent intervals, lead II of the electrocardiogram was repeated and the five leads taken at the time of maximum clinical effect. The latter was designated as one plus when slight ptosis or head palsy was present, two plus if these were moderate, three plus if they were complete and associated with general flac-

cidity and four plus if signs of respiratory paralysis were evident. In the study of the electrocardiograms, careful measurements were made of the rate, rhythm, A-V and I-V conduction time, Q-T interval, and the amplitude, direction and duration of the individual components of the electrocardiographic complex.

Results—Table I summarizes our results. It will be seen that the clinical muscular effect was in most cases moderate to marked. It reached its peak in two to seven minutes and disappeared in from 12 to 20 minutes.

Five cases (1, 2, 4, 5, and 6 in Table I) had normal hearts. Five others (3, 9, 14, 15, and 20) had minimal clinical cardiac involvement, due to hypertension or syphilis. The other eleven were cases of heart disease with congestive failure, who had been treated with digitalis. The degree of failure is tabulated according to the functional criteria of the American Heart Association, with graduation from the asymptomatic (Class I) to the most severely affected group, dyspneic at rest (Class IV). At least three (7, 11, 18) and possibly two others (17 and 19) were overdigitalized.

No apparent differences in clinical response to the injections of curare were noted in these groups. The dosage used was approximately similar, and the two batches of the drug employed during this study were approximately of equal strength. Some of the greatest curare effects (Cases 9, 11, 18, and 20) occurred in our worst cases of heart disease (11 and 18). Yet these, like all others, showed no electrocardiographic changes from the control curves. Results in cases of auricular fibrillation and flutter, digitalis toxicity, bundle branch block, nodal rhythm, and myocardial and pulmonary infarction differed in no way from those without electrocardiographic evidence of myocardial damage. We chose certain cases of digitalis toxicity, nodal rhythm and other arrhythmias, where a known vagus effect is present, especially for the purpose of demonstrating any possible antivagal effect of curare when employed in clinical dosage. We found none. The rate, rhythm, P-R, QRS and Q-T intervals, and the amplitude and configuration of the individual complexes of the electrocardiogram showed no significant variation in any of the twenty-two cases.

Marked somatic tremor was obliterated by the administration of curare (e.g., Case 4, Fig. 2). There were slight increases in heart rate (Cases 5, 6, 11, and 20) with disappearance of sinus arrhythmia in another case (Case 13). Minor variations in T-wave amplitude were seen in two cases (5 and 7). The conformation of the ST segment showed a minimal change in Case 12. We regard these changes as insignificant. Representative electrocardiograms are shown in Figures 1 and 2, and more data are listed in Table I. The absence of important change after curare renders further illustration superfluous, and these strips of tracing are reproduced primarily for the purpose of showing the variety of cases that we have studied.

Comment—The neuropsychiatrists have frequently ignored evidences of cardiac disease in cases of mental illness urgently requiring shock therapy, and have successfully administered the treatment without mishap. However, warnings have been sounded repeatedly of the danger of electrocardiographic and clinical injury to the heart, and even of cardiac fatalities, from the use of various forms of shock therapy.[1,19,20] The addition of curare to metrazol or electric convulsive therapy immediately raised the question as to whether it could result in unfavorable cardiac effects. Wooley and his co-workers[19] have confirmed Bennett[6] in declaring that the blood pressure and heart action were not seriously affected in any of numerous treatments. Burman[10] has noted no change in pulse or blood pressure.

In a recent study,[18] however, two of a series of twenty cases showed marked "cardiac embarrassment," which was apparently secondary to apnea. Critical examination of these case records offers little positive evidence that these reactions were primarily of cardiac origin; the cardiac manifestations seem more likely to have been secondary to the respiratory difficulties. Williams[9] has reported untoward cardiac effects in three of twenty-one cases in which repeated injections of erythroidine (800-1000 mgs.) and metrazol were administered. One of these cases died, apparently in apnea and shock. The other two cases, middle-aged patients without demonstrable heart disease, survived. One developed coma, apnea and ventricular asystole; he responded to artificial respiration and the intracardiac injection of epinephrine. The third patient showed bradycardia and drop of blood pres-

TABLE I

Case No.	Patient	Color	Sex	Age	Curare Mgs/Kg	Diagnosis	Max. clinical effects: head	eyes	sum-total	Anti-dotes	Electrocardiographic findings
1.	J. L.	W	M	64	1.1	Familial spastic spinal paralysis	—	3+	3+	0	Reg. sinus rhythm. Left axis deviation only.
2.	M. L.	W	M	75	1.1	Familial spastic spinal paralysis	—	2+	2+	0	Reg. sinus rhythm. Normal ECG.
3.	M. P.	N	M	44	1.5	Hypertens., enlarg. heart, Class I. Old cerebrovascular accident.	2+	—	2+	0	Reg. sinus rhythm. T_1 & T_4 neg. T_2 diphasic. ST_2 depressed 1 mm. Def. abnormal ECG.
4.	T. B.	W	M	30	1.4	Transverse myelitis (post-traumatic).	3+ Diplopia, dysphagia, arm palsy, mass reflex gone.	3+	3+	0	Reg. sinus rhythm. T_1 low. Not def. abnormal ECG.
5.	C. B.	W	M	42	1.1	Transverse myelitis (post-traumatic).	1+	2+ Diplopia	2+	0	Sinus arhythmia. T_1 low. T_4 diphasic. Not def. abnormal ECG.
6.	L. McC.	W	F	46	1.4	Transverse myelitis.	3+	2+	3+	0	Sinus arhythmia. T_4 low & T in C_2 F neg. Suggestively abnormal ECG.
7.	B. J.	N	M	53	1.6	Hypertens. & luetic cardiac, enlarg. heart, auric. fibrillat., congest. failure, Class III. Diabetes. Syphilis. Alcoholism. Digitalis toxicity.	2+	1+	1+	0	Nodal rhythm. Multifoc. ectopics. Bundle branch block. Neg. T_4. Def. abnormal ECG.
8.	E. H. T.	N	M	80	1.4	Arterioscl. cardiac, enlarg. heart, auric. fibrillat., congest. failure, Class III. Chronic arthritis. Digitalized.	2+	2+ Diplopia	2+	0	Auric. fibrillat. Rare ventric. ectopics. $T_{1\,2\,4}$ diphasic. $ST_{1\,4}$ depressed.
9.	M. M.	N	F	47	1.4	Luetic aortitis, simple. Atrophic arthritis.	4+ Paralysis body & respiration	4+	4+	Oxygen prostigm. 1 mg I. V.	Reg. sinus rhythm. Normal ECG.
10.	J. R.	N	M	61	1.3	Hypertens. & arterioscl. cardiac, enlarg. heart, congest. failure, Class II. Diabetes. Digitalized.	1+	—	1+	0	P-R interval 0.20-0.21 sec. Reg. sinus. $T_{1\,2\,4}$ low, Def. abnormal ECG.
11.	G. C.	N	F	51	1.4	Hypert. & luetic cardiac, enlarg. heart, aortic insuffic., congest. failure, Class II. C.n.s. lues. Digitalis toxicity.	3+ Grunting, hoarse	2+	3+	Oxygen prostigm. ½ mg I. V.	Nodal rhythm, reciproc. beats $T_{1\,2}$ neg. Def. abnormal ECG.

Case No.	Patient	Color	Sex	Age	Curare Mgs/Kg	Diagnosis	Max. clinical effects: head	eyes	sum-total	Anti-dotes	Electrocardiographic findings
12.	W. S.	W	M	64	1.3	Hypert. & arterioscl. cardiac, enlarg. heart, congest. failure, Class III. Syphilis. Digitalized.	2+	1+	1+	0	Reg. sinus rhythm. Low $T_{1\ 2}$. Diphasic T_4. Depressed $ST_{2\ 4}$. Def. abnormal ECG.
13.	D. M.	N	F	31	1.4	Hypertens. (nephritic) cardiac, enlarg. heart, congest. failure, Class II. Acute nephritis. Digitalized.	2+	1+	1+	0	Reg. sinus rhythm. Neg. $T_{1\ 4}$ diphasic T in C_2 F. Depressed $ST_{1\ 4}$.
14.	M. T.	N	F	55	1.1	Hypertens., slightly enlarg. heart. Class I. Duodenal ulcer.	2+	2+	2+	0	Sinus bradycardia. Normal ECG.
15.	R. H.	N	F	60	1.1	Hypert. & arterioscl. cardiac, enlarg. heart, Class I. Diabetes, Gastroenteritis. Digitalized.	2+	1+	1+	0	Reg. sinus rhythm. Normal ECG.
16.	G. F.	W	M	52	1.3	Hypertens. cardiac, enlarg. heart, congest. failure, Class II. Digitalized.	—	2+	2+	0	Reg. sinus rhythm. T neg. in C_2 F. T_4 diphasic. Def. abnormal ECG.
17.	K. L.	W	M	50	1.25	Arterioscl. cardiac, old myocardial infarct., enlarg. heart, congest. failuse, Class II. Lupus erythematosus. Digitalized.	2+	2+	2+	0	P-R interval 0.23 sec. Reg. sinus rhythm. $T_{2\ 3}$ neg. T_1 low. T_4 diphasic Q_3 2mm. Def. abnormal ECG, old posterobasal infract.
18.	A. G.	N	M	60	1.2	Arterioscl. & hypertens. cardiac, enlarg. heart, pulmonary infarct., parox. BBB, congest. failure, Class IV. Digitalis toxicity.	2+	3+ Dyspnea cough	3+	0	Reg. sinus rhythm. T_1 low, T in C_2 F neg., T_4 diphasic. Def. abnormal ECG.
19.	T. B.	N	M	70	1.1	Hypert. & arterioscl. cardiac, enlarg. heart, auric. flutter, congest. failure, Class III. Diabetes. Digitalized.	2+	1+	2+	0	Auric. Flutter. T_4 neg. Def. abnormal ECG.
20.	J. B.	N	M	38	1.1	Malignant hypertens., enlarg. heart, Class I.	4+	4+ Choking throat	4+	prostigm. ½ mg I. V.	Reg. sinus rhythm. Neg. T_2 Def. abnormal ECG.
21.	A. H.	N	M	60	1.1	Arterioscl. & luetic cardiac, enlarg. heart, aortic insufficiency, congest. failure, Class IV. Syphilis, Digitalized.	2+	2+	2+	0	Reg. sinus rhythm. T_1 diphasic, T_2 neg. Def. abnormal ECG.

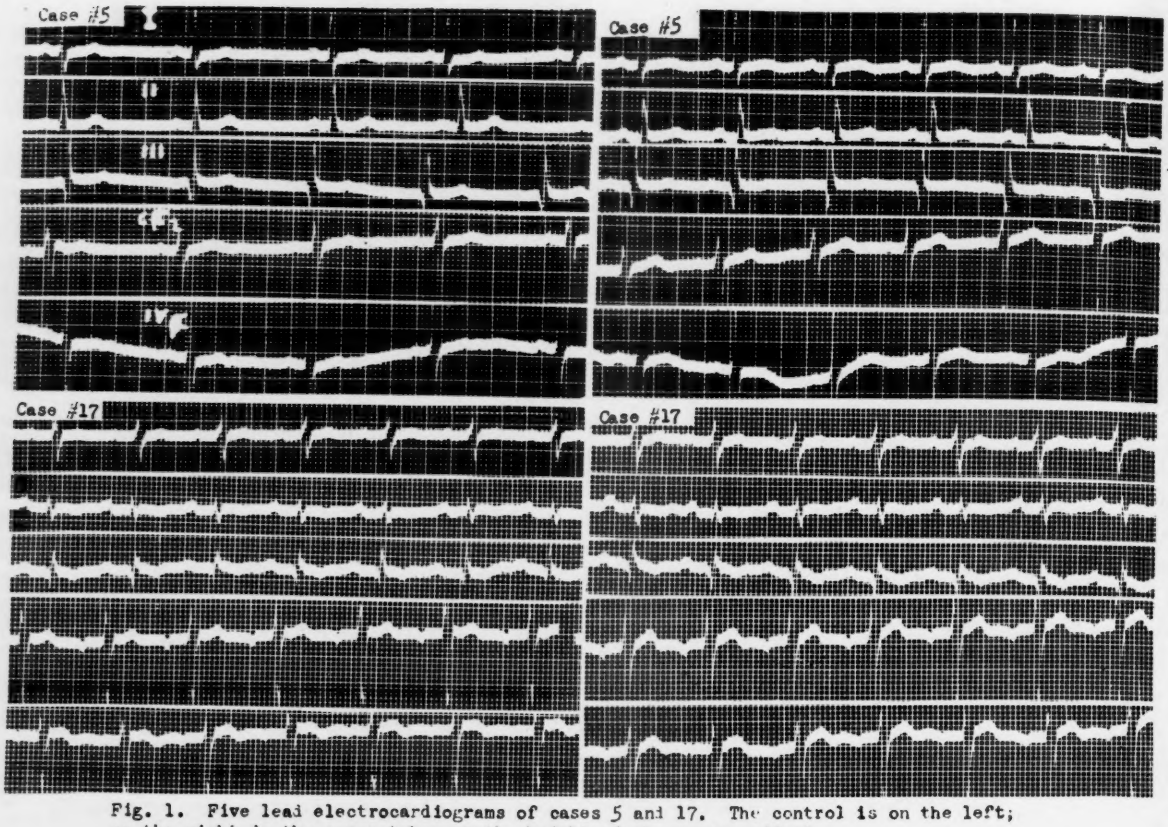

Fig. 1. Five lead electrocardiograms of cases 5 and 17. The control is on the left; on the right is the curve taken at the height of the curare effect.

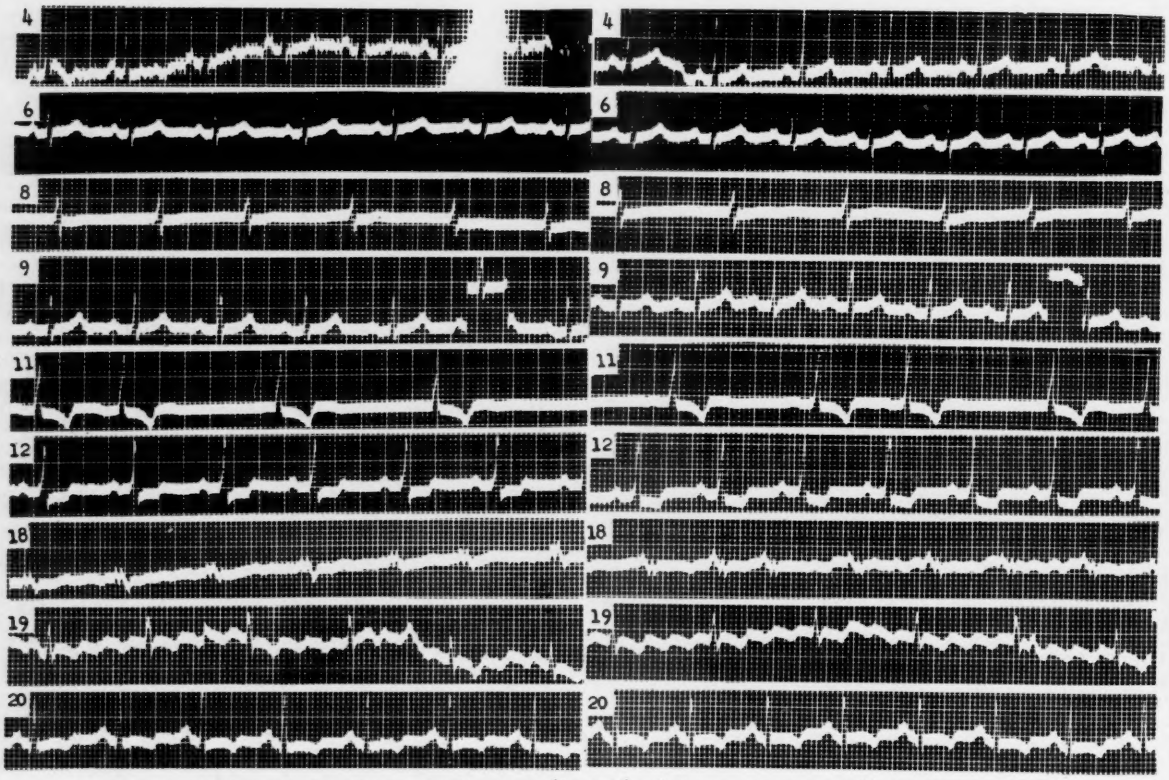

Fig. 2. Strips of the electrocardiograms (lead 2) of 9 cases, representing various normal and abnormal tracings; the control curves are on the left; on the right are those taken at the height of the curare effect.

sure, with neither apnea nor loss of consciousness. He improved following metrazol, epinephrine and caffeine, though the heroic medication produced many auricular premature beats.

It is true, of course, that curare overdosage with apnea and resulting anoxia would tend to aggravate any existing coronary insufficiency and might, theoretically at least, be responsible for sudden death in those cases with previously existing pulmonary congestion due to left ventricular failure. No such fatalities have been reported, but they are not inconceivable, and the possibility should lead us to keep the dose of curare at a minimum and to use it only when prostigmine and oxygen are at hand.

Electrocardiographic studies following the administration of erythroidine alone, as well as of the other curare analogues, are in order. This is especially true since insulin, metrazol and electric shock have known cardiovascular, and not infrequently electrocardiographic, effects.[1]

Our own results confirm in man the previously reported absence of electrocardiographic changes after curare in animals.[13] They also confirm electrocardiographically the older pharmacological studies[2,5] which showed that vagus paralysis and other cardiovascular effects are not present after therapeutic dosage. Since "Intocostrin" is only one form of curare, and the various alkaloids of curare have different actions, our results can apply to it alone. However, these findings do apply to the clinical use of curare, since we have employed the dosage actually used in practice. We have shown that such dosage is harmless from the electrocardiographic standpoint. Individual differences in susceptibility of patients to the paralytic effects of the drug are apparently great, but electrocardiographic effects are absent, regardless of the clinical muscular response, or moderate degree of temporary asphyxia.

Conclusions

1) Employed in clinical dosage, curare exerts no electrocardiographically manifest effect upon the heart.

Author's Note: Acknowledgment is made to E. R. Squibb & Sons, New York, for supplies of "Intocostrin," the form of curare used in these studies.

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Psychosomatic Medicine

Introductory Remarks

SOLOMON KATZENELBOGEN, M.D.*
Washington, D. C.

While the term "psychosomatic" is relatively new, it is interesting to note that the very essence of the meaning and scope of present-day psychosomatic medicine was precisely and unequivocally expressed in ancient time. In the Dialogues of Plato, the Tharacian King Zamolxis says: "* * * this is the reason why the cure of many diseases is unknown to the physicians of Hellas, because they are ignorant of the whole which ought to be studied also; for a part can never be well unless the whole is well * * *. This is the great error of our day in the treatment of the human body, that physicians separate the soul from the body."

The term "psychosomatic" serves the purpose of reminding us of the ever-present connections between bodily and mental functioning. The automatism prevalent in the somatic functions which are subject to our will, such as those of locomotion and speech, is apt to becloud the fact that they are both caused and perceived by mental functioning. The functioning of the vegetative organs, under normal conditions of emotional equilibrium, appear to be uninfluenced by mental functioning. However, the effect of perturbed emotions—of unpleasant or pleasant nature—on the function of the heart, vessels, respiration, digestion, elimination, and on the endocrine and metabolic functions is well known.

On the other hand, pathological changes in the function of both somatic and endocrino-vegetative organs have their reverberations in the mental functioning. Bedside observations of our ancient predecessors originated such maxims as "A sound mind in a sound body," or "Diseases of the organs above the diaphragm are apt to be associated with euphoria, and diseases below the diaphragm usually cause bad humor."

For didactic and also practical clinical purposes, the teaching of psychosomatic medicine should cover at least the following six aspects:

1) The relationship between mental and bodily functions may be primarily a mutual relationship of cause and effect. Abnormal personality reactions may provoke abnormal function of organs, and disturbed organic function may cause mental disorders.

2) Both "psyche" and "soma" may contribute more or less to a pathological condition of either.

3) Psychological factors of the nature to cause serious mental conflicts may express themselves under the guise of somatic feelings, signs, and symptoms. The latter are symbols of psychogenic material loaded with emotion, of which the patient may be only faintly aware, or completely unaware.

4) Psychological reactions of the patient toward his illness.

5) The physician-patient relationship.

6) Treatment.

From the emphasis laid heretofore on the ever-present close functional relationship between mental and somatic functioning, the inference may be drawn that the term "psychosomatic," implying, as it were, division of the organism into "mind" and "body," is not warranted. At a closer examination one can readily see, however, that the suggested dichotomy with its corollaries not only rests on a sound factual basis but is also fruitful for practical clinical purposes: Firstly, the fact remains that there are diseases primarily of a physical nature, and those primarily of a mental nature. Secondly, the term "psychosomatic" brings forward the existence of the above-described relationships between mental and bodily functions. Thirdly, granting that somatic and mental functioning represent only two aspects of the functioning of the organism as a whole, one also recognizes the necessity of concentrating, as the particular case requires, on the study of either the somatic or the mental functioning.

In continuing with definitions, the terms "organic" and "functional" should be considered. There is no need here to dwell upon the meaning of organic diseases. Studies in anatomy, physiology, chemistry, and clinical studies of patients in the general hospital wards make the student undoubtedly feel that

*Clinical Professor of Psychiatry, George Washington University, School of Medicine.

a real physical disease cannot be anything else than an organic disease. Any complaint by the patient or even any obvious disorders in the function of organs in a patient in whom the so-called objective clinical and laboratory studies have not revealed anything abnormal are regarded as neurotic disturbances which to many a physician are synonymous with imaginary maladies. It is, of course, axiomatic that any function has a material background.

It does not follow, however, that the inability to detect structural disorders should lead one to questioning or minimizing the validity of the patient's complaints or to doubting the existence of genuine disorders in the function of organs.

To begin with, the non-detection of structural disorders does not necessarily exclude their presence. In the field of neurology, for instance, chorea, athetosis, Parkinsonism and Thompson disease were transferred in the last three or four decades from the field of functional disorders to that of organic diseases. Furthermore, clinical and anatomo-pathological studies in the domain of general medicine have shown these striking facts: In patients who did have typical anginal attacks the autopsy findings of the myocardial vessels and of the autonomic nervous system proved to be negative. On the other hand, in certain individuals who during their life have never suffered from anginal attacks, the autopsy revealed sclerosis of the coronary arteries. One also knows that asthma may be not only of organic or allergic origin but also of so-called functional nature in which psychological factors obviously play a role. In case of cerebral arteriosclerosis there is no consistent relationship between the severity of vascular sclerosis, as revealed by autopsy, and the sensorium disturbances during the patient's life. No memory disturbances, or very mild ones, were present in individuals in whom the post mortem examination showed quite marked vascular sclerosis. In other cases of so-called functional somatic disorders, the reverse was found to be true. Thus, in certain cases of hyperchlorhydria, of increased peristalsis of the gastro-intestinal tract, of spasms of any of the cavitary organs, of tachycardia, of respiratory disturbances, of metabolic disorders the changes may be reversible. In these cases of functional disorders in which psychological factors are potent, etiologic, and pathogenic agents, there are definite structural and physico-chemical changes in the tissues. These changes are just as much organic as those of so-called organic diseases. The only difference is that while structural alterations as found in organic diseases are more or less irreversible, though apt to undergo transformations, and therefore can be detected, the changes just described occurring in functional disorders are not only reversible but extremely labile, subject to rapid fluctuations and are likely to disappear at one examination or another. Often, the nature of the change is such (osmotic pressure and other physico-chemical changes) that it cannot readily be elicited.

These few instances warrant the following conclusions:

1) There is no consistent relationship of cause and effect between structural disorders and abnormal functioning of organs.

2) No hard and fast line can be drawn between organic and functional disorders.

3) There should be no room for the term and concept of "imaginary malady." The patient's complains and feelings are realities, whether or not organic structural modifications can be elicited.

To state briefly the essential points of our discussion and the inferences to be drawn therefrom, it may be said that the essence of traditional organic-structural medicine is to focus the attention on the function of organs and to recognize as worthy of medical attention and care only those pathological conditions which have a background in structural disorders which can be elicited. The growth of our knowledge in medical and related physical, chemical, and other natural sciences makes it impossible for any one individual to master such an ever-growing accumulation of scientific data.

This unavoidably leads to specialization. The tremendous advantages of specialization are self-evident and connot be too strongly stressed. It is responsible for the comprehensive and detailed studies limited to individual organs or systems of organs. It thus contributes to scientific advancement. By the same token, the specialist is able to contribute effectively to the diagnosis and treatment. However, these desirable features, also carry with them unfavorable elements. The latter find picturesque description in the statement

of Leube which epitomizes the quintescence of the attitude of traditional medicine. "Long questioning of the patient is waste of time. The diagnosis should result like an experiment in physics and chemistry from objective findings."

Those who follow Leube's precept leave out of consideration the carrier of the diseased organs, that is, the patient as a person. Yet, the patient's emotional reactions and behavior in many a case of somatic disorders prove to be very potent, etiologic, and pathogenetic factors. Thus, psychosomatic medicine in using all the knowledge which traditional medicine can offer also makes practical use of the evidences regarding the multifarious relationships between physical disorders and mental reactions. To use McFee Campbell's very apt terms, the exclusively "impersonal medicine"—formerly the pride of Leube's traditional medicine—is being supplemented by "personal medicine." The latter makes it obligatory to include into medical curriculum the study of personality function.

The term personality is derived from the word "persona," which originally meant the theatrical mask used in Greek drama. Certain modern definitions of personality function follow the patterns of the ancients in that they are concerned exclusively with the social effectiveness of the individual, that is, with personality traits and actions which account for success or failure of the individual in his contact with others. The weakness of such sociological definitions, obviously, lies in their leaving out of consideration the subjective, i.e., the inner life of the individual; his ideas, concepts, joys, sorrows, worries; these and his relationship with the external world constitute the full life of the individual. Thus, taking into consideration that both social effectiveness and the inner life constitute integral parts of the function of the person, the common sense definition of personality is the individual as he or she is and acts under various circumstances.

To satisfy the needs of both "Impersonal" and "Personal" medicine, the most effective and practical approach in everyday medical practice is to be found in Adolf Meyer's psychobiology or objective psychology. The dictionary by Hinsie and Schatzky reveals that the term "psychobiology" was used in 1886 by Bernheim; in 1909 by Charles Henry in a book entitled "Psychobiologie et Energetique"; and in 1912 by Oskar Kohnstam in "Psychobiologische Abhandlungen."

In this country, the term "psychobiology" was first introduced by Adolf Meyer in 1915. Since then it has been used in medical and, more specifically, psychiatric literature, with various meanings and connotations: "Science which deals with the function of the person as a whole, the function of the person as an integrated unit." Stedman's Dictionary defines "psychobiology" as "the science of personality formation and personality function." The definition by Hinsie and Schatzky is "the study of the biology of the psyche. It includes such subdivisions as the organization, anatomy, physiology, and pathology of the mind." None of these definitions, however, describe fully the scope of psychobiology. Their inadequacy lies in the fact that, in placing emphasis either on the psyche or the organism as a unit, they are narrowing its actual field. For, psychobiology concerns itself not only with functions regarded essentially as functions of the person as a whole, but equally with functions of organs, systems of organs, with all the physical, chemical, and metabolic processes going on in a living organism. Thus, to do justice to the meaning and scope of psychobiology, a more comprehensive definition is needed. Such was provided by Dr. Meyer in a statement he made to a class of students, to-wit: "Psychobiology studies not only the person as a whole, as a unit, but also the whole of man." The definition implies that psychobiology considers as its domain both the psychology and physiology of the normal and abnormal person. It thus includes into its field of study biological and medical sciences dealing with part functions of the organism, i.e., the function of organs and their metabolic processes; the function of the person as he or she is, and the mutual interrelationship between the function of the person and that of his or her organs.

In brief, the psychobiological attitude in psychosomatic medicine makes it necessary to study the complaints of the patient and objective signs and symptoms not only in terms of physiology and pathology of functions of organs but equally in terms of personality function. Inasmuch as the mode of reactions of a person is determined not only by the immediate environment and events he

is to cope with, but equally by his past experiences and pattern of reactions established throughout his life, the personality study in psychosomatic disorders is as essential as the physical examination. However, the examiner's aim should be not merely to collect numerous facts with the object of obtaining a full biography of the patient, from his very early childhood and throughout his adult life, but to bring forward those facts, which, in his judgment, might throw light on the patient's present difficulties.

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE 83606

Presentation of Case

A 35-year-old white man was admitted to the Neurological Service of the Cincinnati General Hospital on December 13. The only history obtained was that of heavy drinking for one week at least, followed by a blow to the head about November 26. He had complained of headache since the injury, but his appetite had been good. In the two weeks before his admission to the hospital he had had fever on several occasions, which ranged between 101.8 and 102, but for the most part his temperature had remained below 99.5 degrees F. This data was furnished to us by a senior medical student who was in medical charge of the Transient Service Bureau, where the patient had resided since the injury. The patient first appeared at the Transient Service Bureau after the blow on the head, and there gave the story as noted. No relatives were found and it was not possible to elucidate the history at any time after the hospital admission. The student reported to the hospital that he had observed delayed response to questioning, dullness and confusion. The only other abnormality reported before his admission was a gradually increasing stiffness of the neck. Because of increasing stupor, which at times alternated with relatively rational behavior, he was sent to the hospital where he remained until his death on March 7. It was noted by the Transient Service Bureau that he was thought to have received a blow on the left side of the head with a club or blackjack before he entered into their care. It is not known whether or not he was unconscious.

At the initial examination of December 13, the following vital signs were recorded: T. 99; P. 96; R. 26; and B. P. 120/80. There was early swelling of the optic discs. The visual fields were full to confrontation tests, the pupils were normal. No abnormalities of the cranial nerves were found. The neck was stiff. No abnormalities were noted in the motor examination with the exception that all movements were slowly performed. He required considerable urging before performing any test. Passive movement was resisted by the patient. There were no abnormalities in a thorough examination of the reflexes and he appreciated pinprick throughout. Further examination of sensation was impossible.

It was difficult to determine whether or not the patient was suffering from dysphasia. He reached for a pencil with his right hand, and in response to a request to write his name wrote something that was reasonably correct, although he repeated the last syllable several times. He finally repeated his last name and again tacked on the last syllable several times. Seemingly he would have continued to write unless he were requested to desist. He called a pencil and a pen, a "thill." He obeyed simple commands slowly and correctly, but he would either repeat the movement over and over, or he would freeze in the eventual position, maintaining it for a minute or so. He repeated certain sounds over and over. He was never

oriented, and his speech usually was unintelligible. He was apathetic and for the most part unable to cooperate during his hospital stay.

Cerebrospinal fluid: From December 13 until March 5 he had 47 lumbar punctures. The first fluid revealed an initial pressure of 150 mm. Removal of 7 cc. of CSF reduced the pressure to 130 mm. The fluid resembled ground glass and contained a lace-like clot. There were 600 white blood cells per cmm., 68 per cent of which were polymorphonuclear leukocytes, the protein was 210 mg., the sugar 10 mg., and the chloride 594 mg. per 100 cc. Four hours later the pressure was 250 mm., and the removal of 20 cc. of CSF reduced it to 150 mm. The appearance of the fluid was similar to that obtained at the first tap. There were 1500 white blood cells, 80 per cent of which were polymorphonuclear leukocytes. The Wassermann was negative, and the gold curve was 0000122100. In the earlier analyses of the CSF the white blood cells remained around 1000 per cm. The cell counts tended to decrease slowly over the months—on March 1 there were 108, on March 2, 637, on March 5, 180 white cells per cmm. The cells in these latter three fluids were all lymphocytes. Total protein content of the CSF always remained somewhere between 200 and 350 mg. per 100 cc. The sugar content fluctuated tremendously (probably indicating that some fluids were allowed to stand without preservative for some time before sugar determinations were done). The sugar content was recorded respectively as follows: 10, 15, 65, 26, 18, 15, 54, 23, 37, 29, 46, 39, 41, 27, 48, 37, 44, 27, 18, 19, 45, 33, 20, 54, 23 mg. per 100 cc. Five CSF Wassermanns were negative and three CSF Meineckes were positive. Three gold curves

Date	*Sequence of tap*	*Color*	*Diagnostic or Therapeutic Manipulation*
12/27	10	Slightly yellow	
1/5, 6	12, 13	Xanthochromic	
1/7	14	Slightly yellow	
1/14, 15	21, 22	Very slightly xanthochromic	
1/16, 17	23, 24	Xanthochromic	
1/18, 19	25, 26		1/20 Left fronto-parietal craniotomy. CSF from left lateral ventricle contained 220 WBC, per cmm., 20% polys.
2/6	38	Slightly bloody	
2/10			Encephalography "CSF bloody"
2/13	40	Orange	
2/16	42	Orange	
2/19	43	Slightly orange	
2/26	44	Slightly yellow	
3/1,2	45, 46	Straw	
3/5	47	Slightly straw	

were run and all showed a mild to moderate mid-zone curve. Sixteen cultures of the CSF were negative. A guinea pig inoculated on February 26 died on March 30 and was negative for tuberculosis. The CSF cultured for yeast (3/1) was negative. The pressure ranged from 150 to 350+ (over top of manometer) mm. of H_2O; the pressure was high in 26 of 47 taps.

Many specimens of CSF were turbid or cloudy, and on 18 occasions there was slight to marked xanthochromia. A note on the xanthochromic CSF follows.

Laboratory Data: Skull roentgenograms taken on December 13 and January 10 revealed no abnormality. Three blood cultures (1/10, 2/1, 2/4) were negative. A single blood Kahn was negative. The red blood count ranged between 5 and 3.2 millions as the disease progressed. The hemoglobin was 13.6 grams. The white count was 12,000 the day after admission, 89 per cent of the cells were polymorphonuclear leukocytes. The white count remained around this figure, on one occasion it reached 19,000, and on a later occasion the white count was 7400. Repeated urine examinations revealed no abnormalities. The icteric index was 8.7 on admission and later 9, the urea nitrogen 10 mg. per 100 cc., CO_2 combining power 39 vol. per cent; and 40 vol. per cent four days after admission. On February 2 the urea nitrogen was 22 mg. per 100 cc, the CO_2 combining power 53 vol. per cent.

Progress: A thorough physical examination revealed no abnormalities except those noted in the nervous system. The confusion remained and later one obtained the impression that the patient did not have dysphasia when he named objects correctly, tending to perseverate. A tremendous lag between question and answer always remained. The patient was apathetic and mostly uncommunicative, he showed tremulous, picking motions of the upper extremities. He continued to resist any passive movement. The neck remained stiff. He could usually be aroused but he would immediately sink again into stupor if he were allowed to remain undisturbed. Sometimes he would note that he had severe headache when he was aroused. On December 14, he began to show a spiking fever, the pulse following, the fever reaching as high as 103.5 degrees F. (rectal). He was given 142 grams of sulfanilamide over 19 days without changing his condition. On December 18 the temperature had reached 103.5 and it remained at about this level until it went to 104.5 on December 20 and 21, then descended to about 101 where it stayed for the remainder of the hospital course until it rose terminally. On January 4 the neurological abnormalities were noted as follows: pupils about 6 mm. in diameter reacted very slightly if at all to flashlight. He had developed a right Hoffmann and a right Chaddock, and an indefinite left Chaddock. He grasped objects with his hands and would not release them. This resistance like that of the other musculature was probably voluntary. On January 18 he read the first word of a sentence nearly correctly, and then repeated this sound until it became jargon. The perseveration of movements noted in the original examination continued. He lost considerable weight during his hospital stay. On December 19 he weighed 126½ pounds (height 68½"); on January 19, 115; and at the time of his death he was extremely emaciated with so much loss of muscle tissue that his appearance was compared to that of a marasmic infant. On January 20, a left fronto-parietal bone flap revealed no abnormality. The ventricle was reached with a needle at a depth of 4 cm.

On February 10, 60 cc. of air was exchanged for a similar amount of bloody CSF fluid via a needle between L-3 and 4. Ventricular dilatation was demonstrated. On February 15 he did not respond except to occasionally shake his head. He appreciated pinprick everywhere. There was papillaedema. The pupils reacted very slowly to light. Muscle wasting was tremendous. He continued to resist passive movement. In early March he developed a fever of 105 to 106 degrees F. (rectal), the pulse rising to 160. He had numerous decubitus ulcers and the respiratory rate ranged between 50 and 60. During the last month of his illness he held his extremities in flexion, was mainly unresponsive and died on March 7 with signs of pneumonia and toxemia. After the patient was pronounced dead CSF was taken from the cistern, and the right and left ventricles by direct tap. The total proteins were 120 mg., 70 mg., and 66 mg. per 100 cc. respectively.

Differential Diagnosis

DR. HOWARD D. McINTYRE: To be con-

sidered first is the blow on the head of November 26 and the admission to the hospital on December 13; an interval of 18 days during which the patient resided at the Transient Service Bureau. The history that is available was supplied by a medical student, and consisted essentially of a story of alcoholism, headache, stupor, stiff neck, and fever. Two diagnoses are suggested thereby, traumatic brain abscess and subdural hematoma with secondary infection.

The initial examination revealed jargon speech; he seemed to have had an aphasia at various stages of his illness. The neologism is suggestive of aphasia. There are, however, no other focal signs. The perseveration and catatonic attitude suggest generalized rather than localized brain involvement.

The initial CSF contained 600 white cells, the protein 210 mg. per cent, sugar 10; the gold curve midzone. This reminds one of tuberculous meningitis. The ground glass fluid and the low sugar and low chloride content of the CSF would suggest tuberculosis as the etiologic agent, which was excluded by guinea pig inoculation. The negative cultures would seem to exclude all pyogenic meningitis. To be considered are yeast or syphilitic meningitis, or basilar meningitis following a skull fracture; or brain abscess. Yeast was not cultured by special means. The persistent high intracranial pressure after the initially normal pressure reading might suggest the formation of a brain abscess. However, abscess seems to have been excluded by exploration and air encephalography, neither of which gave evidence of a space-consuming lesion. The x-rays do not exclude linear basal fracture through the sphenoid which may be followed by meningitis. The increased cellular content of the second CSF would suggest meningitis around the base. We have seen cases of basilar meningitis secondary to infection of the ethmoidal or sphenoidal sinuses with polymorphonuclear leukocytosis in the early fluids; later analyses of CSF indicating chronicity.

I strongly suspect syphilis. Cases of acute luetic meningitis occur with cell counts of 1000 or more, polymorphonuclear leukocytes predominating. However, five negative CSF Wassermann tests would seem to exclude it, although the Wassermann may be negative in syphilitic meningitis. The three positive Meinecke tests I can't comment on; I question their reliability. Low chloride content is not usually seen in luetic meningitis.

There is some information to be gotten from the course in the hospital. The stupor and grasping and groping indicate generalized brain disease. The lack of effect of sulfanilamide would mitigate against the diagnosis of certain infections which usually respond to this drug. The comparison of his appearance to that of a marasmic infant would suggest that this patient might have had an unrecognized malignancy or encephalitic process with metabolic disturbance. The development of sluggish pupils might suggest localization in the midbrain.

In closing, the following possibilities are considered: subdural hematoma, which was certainly ruled out by exploration and air studies; skull fracture with basilar meningitis; brain abscess, ruled out by air studies; tuberculous meningitis, seemingly eliminated by guinea pig inoculation; purulent meningitis, ruled out by culture, and finally encephalitis to bring up a far-fetched idea. Luetic meningitis is still the one thing I can't get out of my mind. I would list virus meningoencephalitis, but no virus studies were made.

I don't know what the diagnosis is in this case, but I fix upon luetic meningitis as most likely.

DR. MILTON ROSENBAUM: One of the most interesting findings in this case, evident from the space it occupies in the protocal, is the abnormal CSF. I think it should be determined whether or not the CSF was septic or aseptic; whether we are dealing with chronic purulent meningitis or whether the cerebrospinal fluid findings were due to abscess or tumor or something like that. If we follow the classical interpretation, this man had aseptic meningitis. Most of the thymolyzed sugar determinations were within the range of low normal which point to the diagnosis of aseptic meningitis. There was some irritative focus within the nervous system. The one thing I would want to know about is blood in the CSF.

DR. CHARLES D. ARING: All the information that is to be had is in the protocal with the exception of the actual count of red blood cells. This point was determined in the first 23 taps. In no instance was there more than

104 red blood cells per cmm. of CSF. The house officer who performed the encephalogram on January 10 recorded that the CSF was bloody. We know nothing more about this particular CSF. We do not know the cellular content or whether or not the CSF was bloody throughout the procedure.

DR. ROSENBAUM: I don't think this information should be taken too lightly. The clinical picture is reminiscent of severe disturbance of the frontal lobes. There were few localizing signs and with blood appearing in the cerebrospinal fluid sporadically one would have to think of leaking aneurysm; and with the blow on the head, traumatic aneurysm.

DR. MAX KASTAN: There are two types of Meinecke reaction, one reliable and the other completely unreliable. Which of these tests was used?

DR. ARING: Ours was unreliable and has been discontinued. Any sediment in the CSF gave a positive Meinecke test in our laboratory.

Clinical Diagnosis

Acute meningitis changing to subacute over the months, etiology unknown. ? Brain abscess.

Dr. McIntyre's Diagnosis

Meningovascular syphilis.

Anatomical Diagnoses

Aneurysm of the basilar artery (repeated leakage into the interpeduncular space).

Subacute leptomeningitis, due to blood.

Meningovascular syphilis with endangeitis obliterans syphilitica (Heubner type).

Chronic plastic leptomeningitis.

(Early miliary abscesses of liver, kidney, and lungs. Multiple decubitus ulceration. Early lobular pneumonia.)

Discussion of Pathology

DR. I. MARK SCHEINKER: The examination of the central nervous system revealed two gross changes—a large amount of blood between the optic chiasm and interpeduncular space, adjacent to an arterial dilatation which was suspicious of aneurysm; and secondly, a chronic plastic meningitis about the brain stem. The remaining leptomeninges were slightly thickened generally. The ventricular system was markedly dilated. The ependyma was smooth. Histological analysis revealed besides the blood, some of which was fresh and some organized, an aneurysm at the anterior extent of the basilar artery at the point of its division into the posterior cerebral arteries.

There was a comparatively acute reaction in the meninges, surrounding the aneurysm. These leptomeninges were extremely cellular and contained large masses of lymphocytes, plasma cells, and polymorphonuclear leukocytes. Over the dorsal portions of the brain stem particularly there was a chronic plastic reaction consisting of large collections of fibroblasts, and macrophages. The formation of mesenchymal fibers was exceptionally heavy, there was no evidence of activity of the process in this location. The blood vessels were thickened throughout the central nervous system and were partly occluded by a marked proliferation of the intima.

How can these processes be linked together? —on one hand there was relatively acute meningitis, hemorrhage, and aneurysm; on the other chronic plastic leptomeningitis which certainly had been present for many years. The answer to this question is suggested by the lesions in the vessels, a change supposed to be specific of syphilis. We know that a cerebral aneurysm may rarely be associated with syphilis, and that endangeitis obliterans may be responsible for its formation. If this were so in this instance, it would clarify the situation considerably. We would then be dealing with a chronic syphilitic meningitis and endangeitis of the vessels, and subsequently with the formation of a syphilitic aneurysm. With the trauma the aneurysm may have ruptured and produced a hemorrhage into the leptomeninges, associated with which there was an acute leptomeningitis.

DR. McINTYRE: Do you explain the rather acute meningitis on the leaking aneurysm? Why didn't the patient have more blood in the CSF when he was first seen at the hospital?

DR. ARING: The patient was first tapped eighteen days after the supposed onset. Blood could have cleared from the CSF in that length of time. The CSF findings are indicative of repeated hemorrhage.

DR. NATHAN SHAPIRO: Was evidence of syphilis found elsewhere in the body?

DR. SCHEINKER: No.

DR. MARION BLANKENHORN: What is the mechanism of the hydrocephalus?

DR. ARING: In the x-rays taken after the injection of 60 cc. of air, the gas did not get beyond the interpeduncular cistern. There is no air whatsoever over the cortex. In other words, the air was blocked by the tremendous acute and somewhat plastic meningitis at the base, and presumably so was the circulation of the CSF. There is considerable diagnostic evidence in the air studies as far as localization is concerned. One can, I think, see the anterior extent of the lesion at the base, outlined by a semicircle of air.

DR. SHAPIRO: I get the impression that syphilis is undoubtedly responsible. Is that so?

DR. SCHEINKER: Heubner's endangeitis is not present in all cases of syphilis of the central nervous system. I think the evidence at present is that this type of vascular disease has not been proved in cases without syphilis.

DR. SHAPIRO: Are perivascular collections of lymphocytes necessary to make this diagnosis?

DR. SCHEINKER: They are present here. Syphilitic endangeitis is usually associated with perivascular accumulations of lymphocytes.

DR. ROSENBAUM: In the chronic types of meningeal syphilis, the Wassermann reaction is positive in the CSF in only 59 per cent of cases, and in the blood in about 80 per cent.

DR. RICHARD VILTER: Were there no red cells in the CSF examination?

DR. ARING: There were never more than 104 red blood cells per cmm. in the 23 instances in which they were counted. The count usually ranged between zero and 30 red blood cells.

DR. VILTER: Is that due to the fact that the hemorrhage didn't spread?

DR. ARING: You might satisfactorily blame it on the plastic meningitis, the hemorrhage may have been trapped at the base.

DR. SCHEINKER: I think that is a reasonable explanation. There was so much thickening of the leptomeninges that it is difficult to imagine how the blood could spread through this barrier.

DR. ARING: One must remember that on one occasion it evidently did spread.

DR. GEORGE ENGEL: How often in plastic or in acute meningitis do you see xanthochromic CSF?

DR. ARING: I should think practically never if the protein is under 100 mg. per cent. If the protein is over 100, there is usually a slight yellow tinge to the fluid.

DR. ENGEL: Then the CSF findings are incompatible with the diagnosis of meningitis only?

DR. ARING: The supernatant fluid is usually slightly yellow in acute meningitis. However, the information derived from the CSF in this case should have led directly to the diagnosis of repetitive leaking of blood into the CSF. Where the history is deficient as it was on this instance, or even where it is not deficient, it is important to know that one can lean rather heavily on the information gained from dependably studied CSF. This important clinical point has been stressed particularly by Merritt.

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